THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM COCAINE

Using the Recovery Kernel Framework

Cocaine · Crack Cocaine · Freebase · Coke · Snow · Rock · Benzoylecgonine

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 911 or text 988

COCAINE CAUSES HEART ATTACKS AND STROKES — IN PEOPLE OF ALL AGES.

Chest pain during or after cocaine use is a cardiac emergency. Call 911 immediately.

A Note Before You Begin

Cocaine is the drug the compendium was partly written in the shadow of. The Warrior constitution — the Val/Val COMT brain that runs flat at baseline, that needs intensity to feel alive, that finds in cocaine the exact dopamine signal its rapid-clearing enzyme perpetually denies it — cocaine is the Warrior's drug of choice above almost any other.

This is not a metaphor. The compendium states it directly: the Warrior constitution is drawn to stimulants — cocaine, methamphetamine, high-dose caffeine — and to behavioral stimulation: gambling, risk-taking, extreme sports, sexual novelty. The substance floods the system with dopamine faster than even the fast COMT can clear it, producing the experience of aliveness the baseline state does not provide.

Understanding this is not an excuse. It is the diagnostic foundation of everything that follows. The cocaine did not create a brain deficiency. It exploited one that already existed. Recovery is rebuilding the intensity architecture without the substance — a life calibrated to the Warrior's actual neurobiological requirements, through means that do not risk cardiac arrest at age 32.

Cocaine is architecturally different from methamphetamine in ways that change the recovery approach. Where meth forces dopamine release by reversing transporters, cocaine blocks dopamine, serotonin, AND norepinephrine reuptake simultaneously — the triple reuptake inhibition that produces cocaine's distinctive character. This triple mechanism means the serotonin system is also disrupted alongside

dopamine, and the norepinephrine surge is what drives cocaine's cardiovascular danger. The recovery addresses all three.

And there is the adulteration crisis that no cocaine guide should omit: the majority of cocaine in the current supply is cut with levamisole, a veterinary deworming agent that depletes white blood cells and can be fatal. This is not a theoretical risk. It is documented, prevalent, and underrecognized. It gets its own section in this guide.

Section 0 — Immediate Safety: Read This First

⚠ COCAINE CAUSES MYOCARDIAL INFARCTION (HEART ATTACK) AND STROKE IN YOUNG, HEALTHY PEOPLE WITH NO PRE-EXISTING CARDIAC HISTORY. IF YOU HAVE CHEST PAIN, JAW PAIN, LEFT ARM PAIN, SHORTNESS OF BREATH, OR SUDDEN SEVERE HEADACHE DURING OR AFTER COCAINE USE — CALL 911 IMMEDIATELY. DO NOT WAIT.

The Cardiac Emergency

THE CENTURION — The Kill Mechanism That Is Unique to Cocaine Among All Stimulants

Cocaine produces cardiovascular events through three simultaneous mechanisms that combine to create a cardiac risk profile unlike any other commonly used substance:

Mechanism	What it does to the heart	Why it kills young people
Coronary vasospasm	Cocaine causes intense, sudden contraction of coronary artery smooth muscle — the arteries supplying the heart muscle itself. The artery can clamp shut completely, cutting off blood supply to the heart muscle.	A 25-year-old with completely clean coronary arteries can have a heart attack from cocaine because the spasm itself — not plaque — closes the vessel. The standard risk factors for heart disease are irrelevant. Clean arteries can spasm closed.
Norepinephrine surge	Cocaine's norepinephrine reuptake inhibition produces massive sympathetic nervous system activation: heart rate surge, blood pressure spike, increased cardiac workload.	The heart is suddenly demanded to work at maximum capacity while its blood supply may simultaneously be reduced by vasospasm. This combination — maximum workload, reduced supply — is the myocardial infarction mechanism.
Platelet aggregation	Cocaine increases platelet activation and aggregation — the clotting mechanism. Combined with vasospasm and elevated cardiovascular workload, clot formation at the site of spasm becomes more likely.	The triple mechanism — vasospasm, increased workload, enhanced clotting — means cocaine's cardiac risk is not additive with age. A 28-year-old can have a fatal MI from a single use under the wrong circumstances.

⚠ COCAINE + ALCOHOL = COCAETHYLENE. THE COMBINATION IS MORE CARDIOTOXIC THAN EITHER ALONE AND IS RESPONSIBLE FOR A SIGNIFICANT PROPORTION OF COCAINE DEATHS. NEVER COMBINE.

When cocaine and alcohol are used together, the liver produces cocaethylene — a metabolite that has a longer half-life than cocaine, greater cardiac toxicity, and greater lethality than either substance alone. The common practice of using alcohol alongside cocaine to 'smooth the edge' creates a compound more dangerous than either substance. This is not theoretical pharmacology — it is the mechanism behind a significant proportion of cocaine-related deaths.

Overdose Recognition and Response

- Chest pain, tightness, or pressure — cardiac emergency. Call 911. Do not assume it is anxiety.
- Jaw pain or left arm pain — cardiac emergency. Call 911 immediately.
- Sudden severe headache — possible hemorrhagic stroke. Call 911 immediately.
- Confusion, slurred speech, facial drooping, arm weakness — stroke. Call 911 immediately.
- Seizure — cocaine-induced seizure is a medical emergency. Call 911. Place person in recovery position.
- Hyperthermia (body temperature above 104°F) — cooling measures immediately (ice packs to neck, armpits, groin) and 911.
- Extreme agitation, paranoia, or psychosis — cocaine-induced psychosis. Remove from stimulating environment. Call 911 if violence risk or if person cannot be kept safe.

There is no reversal agent for cocaine overdose equivalent to naloxone for opioids. Treatment is supportive — managing the cardiac, neurological, and temperature consequences while the cocaine clears. Time and supportive care are the treatment.

The Levamisole Crisis — The Adulterant Nobody Talks About

THE CENTURION — The Majority of Cocaine in the Current Supply Contains a Veterinary Drug That Destroys White Blood Cells

Levamisole is an anthelmintic (deworming) medication used in veterinary medicine. It has been detected in the majority of cocaine samples tested in the United States and Europe for over a decade. The reason it is added to cocaine is not well understood — it may enhance cocaine's effects through dopaminergic mechanisms or may simply be a bulking agent from cocaine paste processing.

The danger: levamisole causes agranulocytosis — the depletion or destruction of white blood cells (neutrophils specifically). Without neutrophils, the immune system cannot fight bacterial infection. Agranulocytosis from levamisole-contaminated cocaine can be life-threatening.

⚠ SIGNS OF LEVAMISOLE-INDUCED AGRANULOCYTOSIS: Fever, mouth sores, severe sore throat, or unusual susceptibility to infections. If you use cocaine and develop any of these symptoms — go to an emergency department immediately and tell them you use cocaine. A simple blood test (CBC) will reveal agranulocytosis. It is treatable if caught early and cocaine is stopped.

- Levamisole is present in an estimated 70–80% of cocaine supply in the United States.
- Agranulocytosis occurs in approximately 3–8% of people exposed to levamisole — a significant proportion.
- Symptoms typically appear 1–3 weeks after exposure and may be mistaken for ordinary illness.
- Treatment: stop cocaine immediately and eliminate levamisole exposure. The agranulocytosis typically reverses within 2–3 weeks of cessation. Severe cases require hospitalization and granulocyte colony-stimulating factor (G-CSF) treatment.
- Fentanyl test strips do not detect levamisole. There is currently no widely accessible consumer test for levamisole. The only reliable protection is knowing it is present in most cocaine and monitoring for symptoms.

Section 1 — What Cocaine Did to Your Brain

THE CENTURION — The Triple Block That Lit Up the Warrior Brain

Cocaine is a triple reuptake inhibitor: it simultaneously blocks the dopamine transporter (DAT), the serotonin transporter (SERT), and the norepinephrine transporter (NET). This is what distinguishes it mechanistically from methamphetamine, which primarily forces dopamine release. Cocaine does not force release — it prevents reuptake, allowing dopamine, serotonin, and norepinephrine to accumulate in the synapse and continue signaling long after they normally would.

The result is a simultaneous and intense surge in all three monoamine systems — producing the distinctive cocaine experience: intense euphoria and aliveness (dopamine), social warmth and confidence (serotonin), energy and alertness (norepinephrine). The triple hit is cocaine's signature. And it is why the crash from cocaine is often more brutal and more complete than from any single-system stimulant.

The Three Systems Cocaine Disrupted

System	What cocaine did	What recovery requires
Dopamine (DAT blockade)	Blocked the dopamine transporter — prevented dopamine reuptake, allowing it to accumulate in the synapse for 3–5x longer than normal. The reward signal rang loud and long. D2 receptors downregulated in response to chronic flooding.	D2 receptor upregulation through abstinence and BDNF stimulation. Dopamine precursor support (tyrosine). Natural high-intensity reward architecture. The flat, colorless quality of early cocaine recovery reflects D2 deficit — the receptors that heard the signal are temporarily fewer in number. They come back. Build toward them.
Serotonin (SERT blockade)	Blocked serotonin reuptake — the social warmth, the confidence, the emotional expansiveness of cocaine's social effects are substantially serotonergic. Chronic blockade depletes serotonin tone over time.	Serotonin reconstruction through 5-HTP and 5-MTHF. This is a dimension of cocaine recovery that the stimulant guide (meth) does not address as centrally — because meth's serotonin effects are secondary while cocaine's are primary. The social ease that cocaine provided is partly serotonergic and must be rebuilt biologically.
Norepinephrine (NET blockade)	Blocked norepinephrine reuptake — producing the energy, alertness,	HPA axis normalization. The norepinephrine system's chronic overstimulation by cocaine produces the fatigue and energy deficit of

	focus, and physical power of cocaine's stimulant effect. Also producing the cardiovascular danger: elevated heart rate, blood pressure, and coronary vasospasm.	early recovery as the system rests after prolonged activation. Exercise (which naturally activates norepinephrine in controlled amounts) is the primary norepinephrine rehabilitation tool.
Local anesthetic (sodium channel blockade)	Cocaine is the only commonly abused substance that is also a local anesthetic — blocking sodium channels in nerve membranes. This is cocaine's original medical use and explains the numbness associated with intranasal use.	The nasal damage from chronic cocaine insufflation is partly from direct local anesthetic toxicity to nasal tissues and partly from the vasoconstrictive effects reducing blood supply. Nasal mucosal recovery requires cessation — and ENT evaluation if any structural damage has occurred.

The Binge-Crash Architecture — What Makes Cocaine Different

THE CENTURION — The Short Half-Life That Built the Compulsive Pattern

Cocaine has a plasma half-life of approximately 45–90 minutes. This short duration is what drives the binge pattern that is more characteristic of cocaine than of any other stimulant. Unlike methamphetamine (half-life 10–12 hours), which produces a sustained multi-hour effect, cocaine's effects peak within 15–30 minutes and substantially diminish within an hour.

The brain's response to this cycle: the dopamine flood that the first line produced is followed within 45 minutes by a partial withdrawal — a relative dopamine deficit as the flood clears faster than the D2 receptors can recalibrate. The second line is taken not to get high but to prevent the crash. The third line not to get high but to prevent the crash from the second. And so on, through a night that ends not when the person decides to stop but when the supply runs out.

This compulsive binge pattern is the pharmacokinetic signature of cocaine — built into the molecule's half-life. Understanding it is not an excuse. It is the explanation for why stopping after 'just one line' is biologically difficult in a way that would not apply to a substance with a longer half-life.

The Cocaine Class — Forms, Routes, and Their Differences

Form	Route	Onset / duration	Recovery-specific considerations

Cocaine hydrochloride (powder)	Intranasal (snorting)	3–5 min onset / 20–30 min	Most common form in recreational use. Nasal septum damage (perforation), palate erosion, chronic rhinitis, anosmia (loss of smell). ENT evaluation if chronic use. Levamisole contamination is highest in powder cocaine cut for street sale. Phantom smell of cocaine ('clinical edge') documented in compendium during early recovery — the anticipatory brain preloading the olfactory memory.
Crack cocaine / freebase	Smoked (inhalation)	8–10 sec onset / 10–15 min	The fastest onset of any cocaine route — the rapid surge produces the most intense addiction liability and the most severe binge pattern. Pulmonary complications: 'crack lung' (acute pulmonary hemorrhage), chronic cough, respiratory damage. The extreme brevity (10–15 min duration) drives the most compulsive re-dosing pattern. Social and economic context of crack use is distinct and requires specific community architecture considerations.
Cocaine hydrochloride	Intravenous (IV)	Immediate / 10–20 min	Highest bioavailability — entire dose reaches the brain within seconds. Extreme addiction liability. IV-associated risks: vein damage, endocarditis, HIV/hepatitis C transmission, injection site infections. Infectious disease testing and wound care assessment are medical priorities alongside addiction treatment.
Cocaine hydrochloride	Oral / gum rubbing (traditional)	30–45 min onset / 60+ min	Traditional South American use (coca leaf chewing). Much lower peak plasma levels and addiction liability than other routes. Relevant primarily for understanding that the traditional use of coca leaf is pharmacologically distinct from concentrated cocaine hydrochloride.
Cocaine + alcohol → Cocaethylene	Any route + oral alcohol	Variable — duration extended beyond cocaine alone	The combination creates cocaethylene — longer duration, greater cardiac toxicity, documented higher lethality. This is not two substances being used together. It is the creation of a third, more dangerous compound inside the body. The compounding effect is why cocaine deaths disproportionately involve alcohol.

What Cocaine Was Providing — The Compendium's Warrior Diagnosis

THE CENTURION — The Aliveness That the Baseline Brain Could Not Manufacture

The compendium names it with precision: cocaine produces 'the experience of aliveness the baseline state does not provide' for the Warrior COMT brain. This is not a poetic description. It is the pharmacogenomic reality of a brain with fast COMT (Val/Val) that clears dopamine so rapidly that ordinary life's rewards — achievements, social connection, pleasurable activities — fail to produce the sustained reward signal that the brain is calibrated to require.

The Warrior brain at baseline feels flat. Not depressed exactly — more like the world is slightly less saturated than it should be. Like the signal of reward is present but muted. Like you are watching your own life from slightly behind glass. Cocaine removes the glass. Everything becomes immediate, vivid, electric. The conversation becomes brilliant. The music becomes the most extraordinary music ever heard. The task becomes the most interesting task imaginable.

This is not delusion. This is what adequate dopamine signaling feels like in a brain that has never had enough of it.

Understanding this does not make cocaine safe. It makes the recovery architecture clear: the Warrior brain needs genuine intensity, genuine aliveness, genuine stimulation — built through means that do not cause cardiac arrest. What did cocaine actually provide? Map every function. Build every replacement.

Section 2 — Your Genomic Blueprint

Cocaine has the most precisely defined constitutional genomic profile of any substance in this series. The compendium is unambiguous: the Warrior COMT (Val/Val) brain is cocaine's primary genomic target. The genes below define not just vulnerability but recovery strategy with unusual specificity.

The DNA Testing Panel for Cocaine Recovery

Gene	What it controls	Key variant	Cocaine recovery relevance
COMT	Dopamine, norepinephrine, and epinephrine clearance speed — the foundational constitutional gene	Val158Met (rs4680) — Val/Val (Warrior) most relevant	THE primary cocaine vulnerability gene — the compendium names this explicitly. Val/Val (Warrior) fast COMT: dopamine cleared rapidly at baseline → flat, understimulated baseline → cocaine provides the dopamine flooding that briefly overcomes the rapid clearance. Recovery strategy for Val/Val: build a life of genuine intensity and high-stimulation natural rewards. A calm, quiet life will always feel insufficient for this constitution. Do not design recovery for the person you wish you were. Design it for the brain you have.
DAT1 / SLC6A3	Dopamine transporter — the primary target of cocaine's mechanism	10-repeat VNTR allele (rs28363170)	The dopamine transporter is the molecule cocaine blocks. DAT1 variants affect baseline DAT density and function — people with higher DAT expression (10-repeat allele) have faster baseline dopamine clearance and may experience cocaine's DAT blockade as even more dramatically relieving than for those with lower DAT expression. Also relevant to ADHD — DAT1 10-repeat is associated with ADHD, and cocaine is a known ADHD self-medication pathway.
DRD2 / ANKK1	Dopamine D2 receptor density — the receiver for cocaine's dopamine flood	Taq1A (rs1800497) — A1 allele	A1 allele (reduced D2 receptor density) creates Reward Deficiency Syndrome — the chronically 'not enough' baseline. Combined with Val/Val COMT (fast clearance) AND

			reduced D2 density, cocaine is providing relief for a genuinely severe dopamine deficit. Recovery must build multiple dopamine sources simultaneously — single-source natural reward will always feel insufficient for this double-deficit profile.
SLC6A4 / 5-HTTLPR	Serotonin transporter expression — the secondary cocaine mechanism target	Short/short, long/long, or heterozygous alleles	Cocaine blocks SERT — the serotonin transporter. SLC6A4 variants determine baseline serotonin transporter expression and therefore the magnitude of cocaine's serotonergic effect. Long/long carriers (higher SERT expression) may experience particularly pronounced serotonergic effects from cocaine — the social warmth, confidence, and emotional elevation. Recovery must address serotonin reconstruction alongside dopamine.
MTHFR	Methylation — neurotransmitter synthesis foundation	C677T (rs1801133), A1298C (rs1801131)	MTHFR variants impair dopamine and serotonin synthesis efficiency — compounding the DAT1/COMT/DRD2 deficits that drove cocaine use. 5-MTHF is often transformative in cocaine recovery for MTHFR variant carriers because it addresses a genuine synthesis deficit that other supplements do not reach. The flat, colorless baseline of early recovery may be partly a methylation-deficit state rather than purely a withdrawal state.
DRD4	Novelty seeking, dopamine sensitivity, response to reward	7-repeat allele (rs1800955)	DRD4 7-repeat carriers are novelty-seeking, impulsive, and reward-sensitive. This variant is significantly overrepresented in stimulant use disorder and in ADHD. If DRD4 7-repeat is present alongside Val/Val COMT and DRD2 A1, the Warrior constitution's need for novelty and intensity is amplified. Recovery architecture must incorporate genuine novelty, challenge, and unpredictable reward — structured boredom will be intolerable.
CYP3A4	Primary metabolizer of cocaine in the liver	Inducers vs. inhibitors	CYP3A4 is the primary hepatic metabolite for cocaine. CYP3A4 inhibitors (fluconazole, grapefruit, certain antibiotics) can prolong cocaine's plasma half-life and amplify

			its effects. CYP3A4 inducers accelerate clearance. Also relevant: cocaethylene is formed when cocaine and alcohol co-occur — the liver's ethanol-metabolizing enzymes interact with cocaine metabolism to produce this more toxic metabolite.
BDNF	Neuroplasticity — the repair mechanism for cocaine-damaged dopamine circuits	Val66Met (rs6265)	Cocaine directly suppresses BDNF in the prefrontal cortex and hippocampus — one of its primary mechanisms of cognitive impairment. Met carriers have reduced baseline BDNF secretion, compounding cocaine's BDNF suppression. Exercise becomes non-optional for Met carriers because it is the primary available tool for generating the BDNF that cocaine was actively depleting.

Phenotyping Without Test Results

The compendium's phenotyping tools are particularly precise for cocaine because the Warrior profile has a distinctive behavioral fingerprint:

- Did cocaine make you feel like the most alive, most intelligent, most capable version of yourself — not just high but actually yourself at full power? → Strong Warrior COMT (Val/Val) signature. The 'full power' experience is dopamine signaling at the level this brain requires but cannot sustain naturally.
- Did you use cocaine primarily in social contexts — work events, parties, nights out — and feel it enhanced your social performance? → Serotonergic component prominent. SLC6A4 variants may explain particularly strong social effect.
- Did you find the first line never quite enough, always leading to another? → Classic DAT1 10-repeat + DRD2 A1 deficit — the reward system's threshold was raised by downregulation faster than the cocaine could keep up with.
- Have you been diagnosed with or suspected of having ADHD? → DAT1 10-repeat + DRD4 7-repeat association. Cocaine's DAT blockade is structurally similar to methylphenidate's. This may have been self-medication for a genuine ADHD presentation.
- Does the baseline feel not depressed exactly but flat — like the world should be more interesting than it is? → Classic Val/Val COMT Warrior profile. The flat baseline IS the constitution reasserting itself without the chemical. It is real. It is biological. It is the design target for the recovery architecture.

Section 3 — The Detox Protocol

THE CENTURION — The Crash Is the Signal That Recovery Has Begun

Cocaine withdrawal does not produce the seizure risk of alcohol and benzodiazepines. It does not produce the cardiovascular danger of acute cocaine use. What it produces is the opposite: the profound dopamine floor of a system that has been flooded and is now entirely empty.

The compendium names this precisely for stimulants: the system is in low power mode. Nothing feels good or worth doing. The sonic protocol for stimulant withdrawal instructs 60–80 BPM, warm textures, major keys but gentle — because forcing high-energy input into a depleted system produces hollow dissonance rather than activation.

Cocaine withdrawal is not medically dangerous. It is psychologically brutal. And the psychological brutality is directly proportional to how much the cocaine was filling a genuine neurobiological need.

The Withdrawal Timeline

Phase	Biology	Symptoms	What to do
The Crash (Hours 0–24)	Dopamine, serotonin, and norepinephrine reserves depleted; D2 receptors downregulated; reward system at floor; HPA axis exhausted	Profound fatigue and hypersomnia, intense depression and dysphoria, extreme hunger (often for sugar and carbohydrates), anxiety with exhaustion, powerful drug craving	Sleep. Eat. Hydrate. Execute Protocol 01. This is not the time to fight the crash — it is the beginning of repair. The crash is the body's urgent demand for the restoration it was denied during the binge. Allow it.
The Honeymoon (Days 1–3)	Brief partial recovery of dopamine system; some D2 receptor resensitization; HPA axis partially restored; false impression of recovery	Relative improvement after the crash; energy returning; mood lifting; cognitive clarity increasing — this period can feel like 'I'm through the worst of it'	Do not mistake the honeymoon for recovery. This is a partial rebound, not a baseline. The cravings will return, often stronger, in the next phase. Use this window to build the supplement stack, establish the exercise routine, and prepare the environment for the more difficult phase ahead.
The Withdrawal Proper (Days	D2 receptor downregulation stabilizing at new	Persistent anhedonia — inability to feel pleasure from	This is the most dangerous relapse period. The high is remembered; the flatness of

3–28)	lower baseline; dopamine synthesis recovering but not yet at pre-cocaine levels; BDNF suppressed; anhedonia dominant	activities that previously generated reward; intense cravings triggered by cues; depression; cognitive impairment; sleep disruption; irritability; fatigue	recovery is present; the logic of 'one more line just to feel something' is compelling. Environmental redesign (Protocol 05) is critical. Full supplement protocol running. Exercise begins — BDNF is the repair signal. Support structure essential.
Early Recovery (Months 1–3)	D2 receptor upregulation measurably progressing; BDNF recovering; dopamine synthesis normalizing; serotonin system rebuilding; HPA axis stabilizing	Gradual improvement in emotional range; genuine moments of pleasure returning; cognitive clarity progressively improving; PAWS episodes likely; cravings episodic and cue-triggered rather than constant	PAWS episodes for cocaine follow the pattern of sudden, intense cravings (often triggered by specific environmental cues) with accompanying dopamine-deficit dysphoria. Document triggers meticulously. Natural reward architecture becomes increasingly effective as D2 receptors recover.
Consolidation (Months 3–12)	Significant D2 receptor recovery; dopamine and serotonin systems largely normalized; BDNF at or above baseline with consistent exercise; cognitive function recovered	Near-normal hedonic capacity; episodic cravings primarily contextual; genuine enjoyment of life accessible; motivational drive returning; identity reconstruction underway	The natural addiction architecture (Section 6) becomes fully operational. The Warrior brain — with the right environment — finds the genuine intensity it always needed. The compendium's C-28 recast: the Olympian of Nothing is becoming the Olympian of something real.

PAWS in Cocaine Recovery

⚠ COCAINE PAWS IS CHARACTERIZED BY SUDDEN, INTENSE CRAVING OFTEN TRIGGERED BY SPECIFIC CUES — SOUNDS, SMELLS, PLACES, PEOPLE. THIS IS CUE-REACTIVITY AND IT IS POWERFUL. HAVE THE INTERRUPT PROTOCOL READY BEFORE IT ARRIVES.

Post-acute withdrawal syndrome in cocaine recovery is dominated by cue-triggered craving — one of the most powerful cue-reactivity profiles in addiction medicine. The dopamine system was trained to respond to cocaine-associated cues with intense anticipatory firing. Months into recovery, a specific song, a specific social context, the smell of a specific environment can produce a craving state that feels indistinguishable from early withdrawal.

This is not relapse. It is the conditioned dopamine circuit firing from its training, not from current use. The response is the interrupt protocol in Section 5, executed immediately and without negotiation.

Section 4 — Biological Reconstruction

Cocaine recovery requires reconstruction of three systems simultaneously: dopamine (the reward and motivation substrate), serotonin (the social warmth and mood baseline), and norepinephrine (the energy and alertness system). The triple reuptake inhibition that cocaine deployed is met with a triple reconstruction strategy. The methylation foundation underlies all three. Medical recovery from physical complications (cardiac, nasal, infectious) runs alongside the neurological reconstruction.

4.1 — The Methylation Foundation

The triple monoamine depletion from chronic cocaine use is compounded by MTHFR-variant methylation impairment where present. Without adequate methylated folate and B12, the synthesis pathways for dopamine, serotonin, and norepinephrine cannot operate at full capacity regardless of what precursors are provided.

Supplement	Dose	Timing	Cocaine-specific notes
5-MTHF (L-methylfolate)	400–1000mcg daily	Morning with food	Cocaine's triple monoamine depletion requires maximally efficient neurotransmitter synthesis pathways. 5-MTHF is the rate-limiting cofactor for all three monoamine systems simultaneously. For MTHFR variant carriers whose cocaine use was partly self-medication for underlying synthesis deficits, 5-MTHF is often the most impactful single supplement in early recovery. Start at 400mcg and increase slowly.
Methylcobalamin (B12)	1000–2000mcg	Morning, sublingual	Synergistic with 5-MTHF. Cocaine use and the associated lifestyle factors (poor nutrition, alcohol co-use, sleep deprivation) reliably deplete B12. Cocaine's norepinephrine effects place significant demand on the methylation cycle — ensuring adequate B12 supports both the methylation cycle and the nervous system repair.
P5P (active B6)	25–50mg	Morning	Cofactor for dopamine synthesis

			(DOPA decarboxylase), serotonin synthesis (aromatic amino acid decarboxylase), and norepinephrine synthesis (dopamine beta-hydroxylase). Cocaine's triple system depletion places unusually high demand on P5P-dependent enzymes. Use pyridoxal-5-phosphate form only.
Magnesium glycinate	400–600mg elemental	Morning and evening	Cocaine significantly depletes magnesium — both directly through metabolic mechanisms and indirectly through the poor nutrition and sleep deprivation of the binge cycle. Magnesium deficiency worsens anxiety, cognitive impairment, cardiovascular risk, and sleep quality — all of which are relevant in cocaine recovery. The anxiety reduction from adequate magnesium is often noticeable within days.
Zinc	20–30mg daily	Evening with food	Zinc is essential for dopamine synthesis and for the function of several enzymes in the monoamine pathway. Cocaine use and the associated nutritional disruption depletes zinc. Also supports immune function — particularly relevant given levamisole's agranulocytosis risk.

4.2 — Dopamine Reconstruction

THE CENTURION — The Warrior Brain's Primary Target — Rebuilding the Signal That Was Never Enough

Dopamine reconstruction is the central biological project of cocaine recovery. The Warrior brain that cleared dopamine too fast before cocaine cleared it even faster during recovery — a double deficit that requires a deliberate, multi-pronged reconstruction strategy.

Supplement	Dose	Timing	Cocaine-specific notes
L-Tyrosine	500–2000mg daily	Morning, away from protein	Direct dopamine precursor. The compendium names tyrosine specifically for Warrior COMT cocaine recovery. Start at 500mg

			and titrate based on response. Take 30 minutes before or 2 hours after protein — amino acid competition for transport limits effectiveness with food. If L-tyrosine causes anxiety (rare but possible), switch to NALT (N-acetyl L-tyrosine) which has gentler onset.
Mucuna pruriens (L-DOPA source)	100–300mg L-DOPA equivalent	Morning, away from protein	Natural L-DOPA source — one step closer to dopamine than tyrosine. The compendium names Mucuna pruriens specifically for Warrior recovery. Contains additional alkaloids that may support mood independently. Do not combine high-dose Mucuna with L-tyrosine — choose one as primary. Mucuna's effects are faster and more pronounced than tyrosine, which some Warrior constitution people prefer.
Uridine monophosphate + DHA + CDP-Choline (Brain Stack)	UMP 250–500mg + DHA 1g+ + CDP-Choline 250mg	Morning together	Synergistically increases D1/D2 receptor synthesis and density — the most evidence-supported approach to receptor upregulation available without prescription. This is directly addressing the D2 downregulation that is the primary pharmacological consequence of chronic cocaine use. The Brain Stack produces slower, structural improvement over weeks-months rather than acute symptomatic relief.
NAC (N-Acetyl Cysteine)	600–1800mg daily in divided doses	Morning and evening with food	Restores glutamate homeostasis in the nucleus accumbens — directly addressing the glutamate-dopamine dysregulation that drives cocaine craving. Multiple clinical trials specifically in cocaine use disorder support NAC for craving reduction. This is one of the highest-evidence supplements for cocaine specifically. Start immediately on stopping cocaine.
Rhodiola rosea	300–600mg	Morning	Dopamine and serotonin

	standardized		modulation; adaptogen; anti-fatigue. Particularly useful weeks 2–8 when anhedonia and motivational deficit are prominent but the precursor stack alone is insufficient. The flatness of early cocaine recovery responds to Rhodiola's gentle dopaminergic support without stimulation.

4.3 — Serotonin Reconstruction

THE CENTURION — The Social Warmth That Cocaine Was Generating — Build It Back

Unlike the methamphetamine guide where serotonin reconstruction is a secondary target, in cocaine recovery it is co-primary. Cocaine's SERT blockade was providing social confidence, emotional warmth, and interpersonal ease that becomes conspicuously absent in recovery. The social withdrawal and interpersonal flatness of early cocaine recovery is substantially serotonergic.

Supplement	Dose	Timing	Cocaine-specific notes
5-HTP	50–150mg	Evening, 30 min before bed	Serotonin precursor — the most direct available supplemental support for serotonin reconstruction. Evening dosing supports both mood and sleep. The social and interpersonal dimension of recovery — rebuilding the comfort in connection that cocaine's serotonergic effect was providing — often improves noticeably within 2–4 weeks of 5-HTP supplementation. Do not combine with SSRIs without physician guidance.
L-Tryptophan	500–2000mg	Evening, away from protein	Alternative or complement to 5-HTP — slower-acting, gentler onset. Some people find tryptophan + low-dose 5-HTP more effective than either alone. Take away from protein for competitive transport reason.
Inositol	4–12g daily (titrate)	Divided with meals	Modulates serotonin receptor sensitivity. Also has evidence for anxiety and OCD — both of which are more common in

			cocaine recovery than in many other substance recoveries. The rumination and obsessive quality of cocaine craving responds particularly well to inositol's serotonin receptor effects.
Omega-3 EPA dominant	2–4g daily (EPA > DHA)	With food, morning	EPA-dominant omega-3 has the strongest evidence for mood and depression — addressing the serotonergic depression of cocaine withdrawal. DHA supports membrane fluidity for receptor recovery. The EPA:DHA ratio matters for mood: 2:1 or 3:1 EPA:DHA is the evidence-supported ratio for depressive symptoms.

4.4 — The ADHD Question

THE CENTURION — The Most Common Unaddressed Driver of Cocaine Use Disorder

Cocaine is a reuptake inhibitor with structural pharmacological similarity to methylphenidate (Ritalin). The same DAT blockade that makes cocaine euphoric in people without ADHD produces genuine cognitive normalization and focus in people with ADHD. Cocaine has been a primary ADHD self-medication pathway for decades — and the ADHD typically remains undiagnosed through years of cocaine use because 'I can focus when I use it' is not a symptom presentation that gets referred to psychiatry.

DAT1 10-repeat and DRD4 7-repeat variants — both associated with cocaine vulnerability — are also among the strongest genetic markers for ADHD. The overlap is not coincidental.

If you used cocaine primarily for focus, productivity, or cognitive performance rather than euphoria — and if you struggle significantly with attention, organization, and task completion when not using — ADHD evaluation is a clinical priority in your recovery. Non-stimulant ADHD treatments (Strattera/atomoxetine, Wellbutrin/bupropion, Intuniv/guanfacine) can address the ADHD without the addiction liability of stimulants in this context. Discuss with an addiction medicine psychiatrist.

4.5 — Physical Recovery — Nasal, Cardiovascular, and Infectious

THE CENTURION — The Body That Was Damaged Alongside the Brain

Nasal damage (intranasal users): Chronic cocaine insufflation causes nasal mucosal atrophy, septum perforation, and in severe cases, palate erosion. ENT evaluation is appropriate for anyone with chronic intranasal use. Saline nasal irrigation (NeilMed or neti pot) daily supports mucosal recovery. Vitamin C and zinc support tissue healing. Full recovery is possible in mild-moderate cases with cessation; structural damage (perforation) may require surgical intervention.

Cardiovascular assessment: Anyone with significant cocaine use history should have a cardiac evaluation: ECG for arrhythmia or evidence of prior ischemia; blood pressure monitoring; echocardiogram if there are any symptoms or significant use history. Cocaine cardiomyopathy (enlarged, weakened heart from chronic sympathetic overactivation) is documented with heavy chronic use and requires cardiology evaluation.

Infectious disease: IV cocaine users: HIV, Hepatitis C, Hepatitis B testing. Infective endocarditis evaluation if any fever, heart murmur, or systemic symptoms. STI testing if cocaine use occurred in sexual contexts (cocaine and sexual disinhibition are strongly associated).

Levamisole monitoring: CBC (complete blood count) at baseline of recovery. If symptoms of agranulocytosis develop (fever, mouth sores, susceptibility to infection) — emergency evaluation. Repeat CBC at 1 and 3 months.

4.6 — Sleep Reconstruction

Cocaine and sleep have a specific relationship: cocaine causes significant insomnia during use (high norepinephrine activation prevents sleep) followed by hypersomnia during crash (the exhausted system demanding repair time). Chronic binge-crash cycles produce severe circadian disruption that persists for weeks into recovery.

- Melatonin: 0.5–1mg sublingual, 30–60 min before target sleep time. The circadian clock has been repeatedly disrupted by binge-crash cycles — melatonin at the correct timing helps reset it.
- Magnesium glycinate: 400mg elemental evening. Addresses both sleep and the anxiety component of early cocaine withdrawal.
- Glycine: 3–5g, 30 min before bed. Core body temperature reduction and NMDA co-agonism supporting sleep architecture.
- 5-HTP: 50–100mg evening. Serotonin reconstruction supports sleep onset and REM architecture.
- Tart cherry extract: 480mg evening. Natural melatonin source; anti-inflammatory.
- L-Theanine: 200mg evening. Reduces the cognitive hyperactivity that persists in early cocaine recovery and prevents sleep onset.

The compendium's stimulant withdrawal sonic protocol for sleep: delta binaural beats (0.5–4 Hz) for sleep induction; 60–70 BPM maximum; ambient, drone, nature

sounds. No sudden dynamics, no jarring transitions. Low volume. The hypersensitive post-cocaine nervous system is destabilized by stimulating audio.

4.7 — Peptide Support Layer

Peptide	Mechanism in cocaine recovery	Timing	Notes
BPC-157	Dopamine and serotonin system modulation; directly counteracts cocaine-induced dopamine depletion in research models; gut-brain axis repair; neuroprotection; supports BDNF expression	Day 1 post-cessation — daily	BPC-157 is the highest-priority peptide in cocaine recovery for its direct dopamine system modulating properties — animal models show BPC-157 specifically counteracting dopamine system disruption from stimulants including cocaine. Oral or subcutaneous. The gut-brain repair is relevant because cocaine use significantly disrupts gut microbiome and the peripheral serotonin production that the gut generates.
Semax	BDNF and NGF upregulation; cognitive enhancement; dopamine and serotonin modulation; prefrontal cortex support — cocaine directly suppresses BDNF in the prefrontal cortex	Week 1 onwards	Cocaine's direct suppression of prefrontal BDNF is one of its primary mechanisms of cognitive impairment. Semax's BDNF-stimulating effects directly oppose this mechanism. The cognitive fog, impaired decision-making, and impulsivity of cocaine recovery respond to Semax's prefrontal support. Intranasal.
Selank	Anxiolytic via GABA and serotonin modulation; reduces anxiety without dependence; serotonergic component directly relevant to cocaine's SERT mechanism	Weeks 1–8 — as needed for anxiety	Selank's serotonergic mechanism makes it particularly relevant for cocaine recovery versus other stimulants — it addresses the serotonin dimension of cocaine's effect alongside the GABAergic anxiety reduction. Non-addictive. Intranasal.

Epitalon	Sleep architecture; telomere protection; pineal pathway; circadian normalization	Weeks 1–4	The circadian disruption from cocaine binge-crash cycles can persist for weeks. Epitalon directly supports the pineal pathway and circadian normalization.
Dihexa	Synaptogenesis; HGF/c-Met pathway; promotes new synaptic connections in prefrontal cortex and hippocampus damaged by cocaine's BDNF suppression	Months 1–3	Cocaine's suppression of BDNF and its downstream synaptic effects can be addressed at the synaptogenesis level by Dihexa. Very potent — start with minimal dose. Limited human data; strong mechanistic rationale for cocaine-induced prefrontal damage.

Section 5 — The Cocaine Craving — Understanding and Interrupting

THE CENTURION — The Fastest, Most Cue-Reactive Craving in the Stimulant Category

Cocaine cravings are among the most intense and most cue-reactive in addiction medicine. The dopamine anticipatory signal that cocaine trained the brain to produce in response to cocaine-associated cues is extraordinary in its power and speed — within milliseconds of encountering a relevant cue (a specific sound, the sight of white powder, a social context associated with use, a specific smell), the dopamine anticipation circuit fires at near-maximal intensity.

This is not a thinking process. It is a pre-cognitive neurological event. The brain has produced the anticipatory dopamine signal before the prefrontal cortex has had time to evaluate it. This is why the 'just don't think about it' approach fails for cocaine: the craving is not initiated by thinking. The thinking comes after.

The interrupt protocol must therefore be behavioral before it is cognitive: the action comes before the analysis.

Cocaine Craving Trigger Map

Trigger type	The neurological event	Interrupt strategy
Environmental cue (highest intensity)	Specific places, people, paraphernalia, smells, music, social contexts — anything consistently paired with cocaine use produces anticipatory dopamine firing before conscious awareness of the craving.	LEAVE THE ENVIRONMENT IMMEDIATELY. Do not negotiate. Do not 'just say goodbye.' Physical exit from the cue environment is the first action, not the last. The cue-reactivity circuit requires the cue environment. Remove yourself from it before executing anything else.
Social / performance context	Work events, parties, social situations where cocaine was previously used to perform — the brain fires anticipatory dopamine in these contexts because it learned that cocaine preceded them.	Pre-plan high-risk social contexts: arrive late and leave early; bring a sober support person; have an exit excuse prepared; execute the supplement stack before the event. The social confidence cocaine provided must be built through other means (Section 6) before high-risk social contexts are comfortable.

Dopamine-deficit craving (flatness)	The anhedonic, flat quality of early recovery produces a specific craving state: 'I just want to feel something.' The motivation is not the cocaine specifically — it is the end of the flatness. Any dopamine signal would do. The cocaine was just the fastest available one.	This craving requires a high-intensity natural dopamine intervention: vigorous exercise (the fastest available natural dopamine hit), cold exposure, competition, creative challenge. Something with enough intensity to provide genuine reward rather than adequate reward. The Warrior constitution's intensity requirement is not negotiable — it must be met.
Social-confidence craving	Approaching a social situation with the feeling that you are insufficient without the drug — the social ease, confidence, and brilliance that cocaine provided is absent, and the sober social baseline feels inadequate.	5-HTP and social exposure practice. The social confidence was partly pharmacological (SERT blockade) and partly behavioral (cocaine users practice high-engagement social performance on cocaine for years). The behavioral component can be rebuilt with deliberate practice in lower-stakes contexts. The serotonin component is supported by 5-HTP and natural serotonin-activating practices.
Phantom smell (documented in compendium)	The compendium specifically names 'the clinical edge of ketamine' and by extension cocaine's distinctive chemical smell as a phantom olfactory experience in early recovery — the anticipatory brain preloading the sensory memory.	The compendium distinguishes two types: anticipatory (cue-triggered, lasts seconds to minutes, accompanied by craving) and biological release (during exercise, from adipose stores releasing cocaine metabolites, accompanied by nausea rather than craving, lasts longer). Both resolve with time and are evidence of recovery working, not failing.

THE COCAINE CRAVING INTERRUPT — 5-MINUTE PROTOCOL

1. BEHAVIORAL RESPONSE FIRST. Before anything else: if a cue is present — leave the environment. If not — change your physical position immediately. Stand up. Walk to a different room. Go outside. Do not execute any other step while remaining in the triggering environment.
2. HIGH-INTENSITY PHYSICAL INTERRUPT. Twenty jumping jacks, a sprint around the block, cold water on the face, or anything that creates an immediate, intense physical sensation. The Warrior constitution's craving fires at high voltage — it requires a high-intensity counter-signal, not a gentle one. This is not optional softness. It is neurobiological calibration.

3. EXECUTE 4-7-8 BREATHING — SIX CYCLES. After the physical interrupt, the parasympathetic system needs activating. The Warrior brain that just experienced a maximum-intensity dopamine anticipation signal needs the vagal brake engaged. Six cycles, not four.
4. TAKE NAC AND TYROSINE. The glutamate homeostasis (NAC) and dopamine precursor (tyrosine) together address the neurochemical urgency underlying the craving. These are not placebos — they are the raw materials for the reward signal the craving is demanding, provided through the synthesis pathway rather than the reuptake inhibition pathway.
5. CONTACT. One text or call to your support structure. The dopamine anticipation circuit was trained in social contexts — cocaine was the social drug. Connection provides a genuine social dopamine signal that partially addresses the circuit's demand. Name what is happening. You do not need rescue. You need signal.
6. EXECUTE THE HIGHEST-INTENSITY NATURAL REWARD AVAILABLE. From Section 6. Whatever is immediately accessible and genuinely intense. A flat, mild activity will not meet the Warrior craving's threshold. It must be real intensity.
7. DOCUMENT. Trigger, type, intensity (0–10), action, outcome. Three weeks of this data reveals your specific craving architecture with a precision that changes your relationship to the cravings themselves.

Section 6 — Porting the Addiction: Building the Warrior's Natural High

THE CENTURION — Centurian Paper C-28 Applied to Cocaine Recovery — The Aliveness the Warrior Brain Requires

The compendium is precise about what the Warrior constitution needs and what cocaine was providing: the experience of aliveness the baseline state does not provide. Not calm. Not relief. Not escape. Aliveness.

The Olympian of Nothing who spent years generating that aliveness through dopamine flooding has exactly the right engine for something extraordinary. The engine that chased cocaine with the intensity it deserves is the same engine that would pursue mastery, competition, service, and achievement with that same intensity — if given the right address.

The natural addiction architecture for cocaine recovery is not a gentler life. It is a more genuine life. One calibrated to the actual intensity requirement of this brain.

The Warrior's Natural Dopamine Architecture

Source	Dopamine intensity	How to implement	For cocaine recovery specifically
High-intensity exercise — especially competitive	Very High — dopamine, norepinephrine, endorphin, BDNF simultaneously	Running, CrossFit, martial arts, competitive sport — daily, 45+ minutes. The intensity must be genuine, not moderate.	For the Warrior constitution, the intensity of exercise matters more than duration. A 20-minute all-out sprint interval session may provide more dopamine signal than 60 minutes of moderate jogging. The nervous system that cocaine was stimulating requires real physical intensity to generate comparable neurochemistry. This is the non-negotiable pillar. Build it first.
Direct competition	Very High — social stakes amplify dopamine 3–5x beyond solo achievement	Competitive sport, martial arts, combat sports, competitive gaming, chess, sales competitions, debate	The dopamine release of winning — of outperforming a specific competitor in a specific moment — is among the most intense natural dopamine events available. For the Warrior who used cocaine as a performance enhancer in social and professional contexts, competition provides the social-stakes dopamine that cocaine

			was delivering. Find a competition. Enter it. The pursuit of the win is the engine cocaine was running.
High-stakes professional performance	High — performance dopamine with social validation	Public speaking, presentations, entrepreneurship, sales, performance arts, leadership	The Warrior brain performs brilliantly under pressure — the Val/Val COMT that clears stress chemicals fast is the stress-resilient, pressure-performing brain. What cocaine was enabling in professional contexts, the recovered Warrior brain can provide through the legitimate preparation and performance that cocaine was shortcutting. The shortcut cost the person their credibility. Building the real capacity produces lasting respect.
Creative mastery with performance	High — especially with audience and stakes	Music performance, comedy, visual art with exhibition, writing with readers, any creative work that has an audience	The creative flow state that cocaine was artificially inducing — the brilliant conversation, the inspired performance — can be accessed through genuine creative mastery and the practice of performing it in front of others. The difference: cocaine's creative state was borrowed and depleting; genuine creative mastery builds on itself and grows.
Cold exposure + exercise combination	Very High — norepinephrine surge + BDNF + endorphin; the combination produces 2–3 hour sustained elevated mood	Cold plunge followed by exercise; or exercise followed by cold shower. Daily where possible.	The combination of cold exposure and vigorous exercise produces a norepinephrine surge comparable to cocaine's NET blockade effect — the alertness, energy, and aliveness — through the norepinephrine axis, without cardiovascular toxicity. For the Warrior brain that found cocaine's norepinephrine component as compelling as the dopamine component, this combination is the highest-yield natural alternative.
Social depth and authentic connection	Moderate-High — oxytocin + endorphin + serotonin	One to two relationships of genuine depth; recovery community; shared high-intensity activity with others;	Cocaine's serotonergic social effect — the sense of genuine connection, brilliance in conversation, feeling genuinely known and knowing others — was partly pharmacological but partly the permission the cocaine

		service in contexts requiring trust	gave to be fully present and engaged. Recovery rebuilds the capacity for genuine social depth without the permission slip. This takes longer than dopamine recovery but is more sustaining.
Service at scale — helping others recover	High — sustained; the most reliable long-term dopamine source for the Warrior	Peer recovery support, sponsorship, mentorship, advocacy, anything where your Warrior capacity directly improves another person's situation	The Warrior brain in cocaine recovery, with its high-functioning, pressure-capable, intensity-seeking profile, is among the most effective recovery coaches that exists for other people who share that profile. The person who has been where the cocaine user is and who has built the life on the other side has something that cannot be bought or faked. Many people in long-term cocaine recovery find their most reliable and most genuine dopamine source in becoming that guide.

The Social Confidence Architecture — Rebuilding Without the Drug

THE CENTURION — The Serotonergic Work That Cocaine Was Doing

The social ease, confidence, and conversational brilliance that cocaine's SERT blockade was providing is one of the dimensions of cocaine use that people discuss least and miss most. The sober social baseline — for many people with cocaine use disorder — feels not just worse than on cocaine but actively inadequate. Conversations feel effortful. The natural wit and connection that cocaine produced feels absent.

This is real, and it is primarily serotonergic. The serotonin system rebuilds with abstinence and with the supports above. But the behavioral component matters equally: the Warrior who has been performing brilliant social engagement while on cocaine has practiced that performance. The skill is real. It just needs practice without the pharmaceutical confidence booster.

The architecture:

- Start smaller: practice authentic social engagement in lower-stakes environments (smaller groups, trusted people, familiar contexts) rather than jumping straight to the high-stakes professional and social events where cocaine was used.
- Preparation instead of chemicals: the confidence that cocaine provided by flooding the serotonin system can be approximated by thorough preparation — knowing the material, having practiced the conversation, having done the

physical exercise that the body needs to feel capable. Prepared Warrior beats pharmaceutical Warrior over time.

- Serotonin supplementation: 5-HTP (50–100mg evening), inositol, and adequate protein — the serotonin reconstruction supplements in Section 4.3 — provide genuine neurochemical support for the social confidence dimension of recovery.
- The recovery community as social training ground: twelve-step, SMART Recovery, or any recovery community provides a uniquely understanding social environment where the mask is optional and the connection is genuine. This is the serotonergic community that the cocaine social scene was approximating chemically.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map

Phase	Protocols	Primary focus	Cocaine-specific notes
Medical Assessment (Days 0–7)	Physician, cardiology if indicated, ENT if nasal damage, infectious disease	Cardiac evaluation; CBC for levamisole; nasal assessment; STI/HIV/HCV testing; ADHD evaluation if indicated	These medical evaluations should happen in the first week of recovery, not months in. Levamisole agranulocytosis can be life-threatening if undetected. Cardiac damage from cocaine use is progressive. The medical assessment is not optional self-care — it is safety.
Biological Foundation (Days 1–14)	Protocol 07, Protocol 09, Protocol 17, Protocol 18	NAC, tyrosine, 5-MTHF, magnesium, 5-HTP, exercise beginning, sleep stack	NAC on Day 1. Tyrosine on Day 1. Magnesium on Day 1. The triple monoamine depletion requires triple reconstruction support from the start. The compendium's stimulant crash sonic protocol (60–80 BPM, warm textures) for the first week. Protocol 07 (nutrition) emphasizes protein — dopamine and serotonin synthesis require amino acid precursors.
Genomic Mapping (Weeks 1–4)	Section 15 (Compendium), COMT assessment, DRD2, DAT1, SLC6A4, MTHFR	COMT constitution; Reward Deficiency Syndrome scoring; methylation status; ADHD genetic markers	COMT Val/Val confirmation is the highest-priority genomic finding — it changes the entire recovery architecture direction. DRD2 A1 allele identifies the double-deficit Warrior who needs the most robust reward architecture. ADHD markers (DAT1, DRD4) may identify a co-occurring condition requiring its own treatment.
Intensity Architecture (Weeks 2–12)	Protocol 03 (exercise), Protocol 04 (community), Section 6 of this guide	High-intensity exercise daily; competition identification; creative practice; social intensity building	The Warrior constitution cannot be told to rest and let recovery happen to it. It must be given a project with genuine intensity. Identify the natural addiction architecture immediately — what will this person's competitive outlet be? What will their creative practice be? What will provide the aliveness they legitimately require? These decisions made in weeks 2–4 determine the long-term sustainability of recovery.

Environmental Architecture (Weeks 2–12)	Protocol 02, Protocol 05, Protocol 08, Protocol 13	Addiction transfer prevention; cue removal; social architecture redesign; trigger mapping	Cocaine cue-reactivity is among the most powerful in addiction medicine. Protocol 05 (environmental redesign) requires thoroughness about the specific social environments, people, and contexts associated with use. The social drug's social environment must be specifically and deliberately redesigned — not just reduced but replaced.
ADHD Treatment (Weeks 2+ if indicated)	Addiction medicine psychiatrist	Non-stimulant ADHD treatment; cognitive scaffolding; executive function support	If ADHD evaluation confirms the diagnosis, non-stimulant treatment (atomoxetine, bupropion, guanfacine, viloxazine) should be initiated alongside the recovery framework. Treating the ADHD reduces the pull back to cocaine's cognitive effects and provides the executive function support that the brain was borrowing cocaine to achieve.
Identity Work (Months 2–12)	Protocol 20, Protocol 21, C-28	The Warrior identity beyond cocaine; professional and social identity reconstruction; purpose	Cocaine use disorder in the Warrior often involves a professional or social identity that was built with cocaine's performance enhancement. 'I can't do my job without it' is a common and real concern. The identity work confronts this directly: what does genuine professional competence look like, built on real capacity rather than pharmacological enhancement?
Long Game (Months 6+)	Protocol 24, Protocol 25, C-28, C-31	Purpose architecture; competition and mastery fully operational; the Warrior in full expression without the chemical	The Warrior constitution in full cocaine recovery — with a vigorous exercise practice, a competitive outlet, a creative practice, genuine social depth, and meaningful work — is one of the highest-functioning recovery profiles that exists. The same intensity that drove the cocaine use is now driving something real. The compendium was partly written for this person.

Section 8 — The Medical Support Layer

Unlike opioids, benzodiazepines, and alcohol, there is no FDA-approved medication specifically for cocaine use disorder. This is a genuine treatment gap — cocaine use disorder causes significant morbidity and mortality, and the absence of effective pharmacological treatment is one of the most significant failures in addiction medicine.

What does exist:

Medications With Evidence for Cocaine Use Disorder

Medication	Evidence and mechanism	Genomic and clinical considerations
Bupropion (Wellbutrin)	Dopamine and norepinephrine reuptake inhibition; modest evidence for cocaine craving reduction; significant evidence for the depression component of cocaine withdrawal	CYP2B6 metabolized — poor metabolizers experience much higher plasma levels at standard doses. Val/Val COMT Warriors may find bupropion provides sufficient dopaminergic stimulation to reduce cocaine craving. Dual function: treats withdrawal depression AND provides some dopaminergic substitution for the Warrior constitution. Also treats ADHD if present.
Modafinil / Armodafinil	Wakefulness-promoting agent with DAT and NET modulating properties; evidence for cocaine craving and cognitive function in early recovery	Non-stimulant (no amphetamine effects) but promotes wakefulness and provides mild dopaminergic support without significant addiction liability. Particularly valuable for the cognitive impairment and fatigue of early cocaine recovery. CYP3A4 metabolized. Discuss with addiction medicine physician.
Naltrexone	Opioid receptor antagonist — reduces the rewarding effect of cocaine through opioid system modulation; modest evidence for cocaine craving in some studies	OPRM1 A118G carriers show strongest response. Some evidence suggests naltrexone may reduce the euphoric component of cocaine use, making relapse less rewarding even when it occurs. Not first-line but may be appropriate in dual-diagnosis cocaine + alcohol use disorder.
GLP-1 agonists (semaglutide,	Dampening of reward 'wanting'	The most significant emerging development in cocaine pharmacotherapy. The same

tirzepatide)	signal through dopamine and reward circuit modulation; 2025–2026 emerging data shows promising reductions in cocaine use	mechanism that has shown 50–60% reduction in alcohol use and emerging data for opioid overdose is now showing preliminary evidence for cocaine. Discuss with physician — metabolic benefit profile may also address some of the physical health consequences of cocaine use.
Disulfiram (Antabuse)	Off-label use for cocaine — inhibits dopamine beta-hydroxylase, reducing norepinephrine synthesis and potentially affecting cocaine reward	Mixed evidence — some studies show craving reduction, others do not. May be more effective in people with alcohol co-use disorder. Requires absolute alcohol abstinence (produces severe reaction with alcohol). ALDH2 variant carriers: particularly potent reaction with alcohol.
Non-stimulant ADHD treatment	If ADHD is confirmed co-occurring — atomoxetine, bupropion, guanfacine, viloxazine; addresses the underlying DAT/DRD4 deficit that cocaine was self-medicating	This is one of the most clinically impactful medical interventions available for cocaine use disorder in ADHD co-occurrence. The person who was using cocaine to focus now has a non-addictive alternative. This removes one of the primary functional drivers of cocaine use. Discuss with addiction medicine psychiatrist.

A Final Word

THE CENTURION — The Warrior Was Never Looking for Escape. It Was Looking for a Fight Worth Having.

The compendium's dedication is for the ones who ran out of time before anyone told them their biology was not their weakness. In the Warrior constitution, the ones who ran out of time were often the most alive people in the room — the most energetic, the most driven, the most intensely present. Cocaine found them because it was calibrated exactly to what they lacked and what they needed.

What they lacked was not character. It was baseline dopamine signal adequate to sustain the experience of aliveness they were capable of and required. What cocaine provided was the signal. What cocaine cost was everything else.

The recovery documented in this guide is not about becoming a calmer, quieter, more moderate person. The Warrior does not become a Sage in recovery. The intensity does not go away. The need for aliveness does not go away. The dopamine clearance rate does not go away.

What goes away is the need to flood the system with a substance that breaks the heart — literally — to get the signal it deserves.

The Warrior in full recovery — exercising at the edge of capacity, competing in something that matters, creating something that did not exist before, building something that helps the people who are still in it — is not a diminished version of the person who was using cocaine. It is the complete version. The one the cocaine was borrowing against.

The fight was always worth having. It just needed the right address.

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Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357

⚠ **HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE**